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Maximum ultrafiltration rates during peritoneal dialysis in rats

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Maximum ultrafiltration rates during peritoneal dialysis in rats. It has been suggested that filtration pressure equilibrium could occur in peritoneal capillaries during peritoneal dialysis with very hypertonic exchanges. Rats were exposed to peritoneal dialysis solutions using 16 ml instillations, 30 minute cycles, and dextrose concentrations from 1.4 to 20 g%. There was a plateau in ultrafiltration per exchange at mean osmotic gradients above 360 mOsm/kg H_2O near 12.5 ml/ex (0.42 ml/min). The findings are also compatible with filtration pressure equilibrium predictions at an effective capillary plasma flow of 0.84 ml/min and a filtration fraction near 50%. Studies with cardiovascular drugs (norepinephrine i.v., nitroprusside i.p., and dobutamine i.v.) showed no effects on the maximum ultrafiltration rates. This might indicate that flow is rather fixed because of known microcirculatory effects of solutions themselves.

Filtration pressure equilibrium may occur in glomerular capillaries of some species [1]. In glomerular capillaries with adequate filtration fractions, increasing oncotic pressure along the capillaries may eventually equal net transcapillary hydraulic–pressure, preventing additional ultrafiltration. Since glomerular filtrate is very low in protein and is an ultrafiltrate of serum, there is little or no contribution to transmembrane osmotic pressure other than plasma protein oncotic–pressure.

In contrast, in peritoneal dialysis, an osmotic agent, usually glucose, is added to the dialysis solution to generate ultrafiltrate. We have previously summarized evidence to support the hypothesis that the major source of the ultrafiltrate is blood from peritoneal capillaries [2–4].

Ronco and colleagues have suggested that ultrafiltration rates per exchange during peritoneal dialysis should be limited at very high mean osmotic gradients by steep increases in plasma oncotic pressure as plasma filtration fraction approaches 50% [5]. Attempts to increase filtration fraction further by increasing mean osmotic pressure would result in little or no increase in filtration rate since oncotic pressure increases sharply with only small increments in filtration fraction. At lower mean osmotic pressures and filtration fractions, ultrafiltration per exchange might be limited by capillary pore area, hydraulic permeability, and/or filtration pressure equilibrium. Increments in mean osmotic gradient would increase ultrafiltration and filtration fractions regardless of which were most limiting as long as filtration fractions were below 40 to 50%.

The main purpose of these studies was to measure ultrafiltration rates in rats undergoing peritoneal dialysis exchanges at fixed instillation volumes and cycle times with progressive increases in dialysis-solution glucose concentration to levels well above those that are used clinically. An ultrafiltration maximum is shown to be demonstrable. Effects of intravenous and intraperitoneal vasoactive agents on this ultrafiltration maximum were assessed. Our findings raise questions about the magnitude of effective, peritoneal capillary blood–flow relative to the peritoneal dialysis process.

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Methods

Studies in rats

The animal model. Sprague–Dawley male rats, 290 to 375 g, were anesthetized with 50 mg/kg of subcutaneous Nembutal solution (Abbott Laboratories, North Chicago, Illinois, USA). Rats were placed supine on a heating pad at 37°C and body temperature was monitored with a rectal temperature probe (Yellow Springs Instruments, Inc., Model 402). The external jugular vein was exposed and cannulated for intravenous administration. Blood pressure was monitored through a cannula in the femoral artery with a pressure transducer (P 23/D, Gould Statham, Hato Rey, Puerto Rico) connected to a polygraph (Low–Level D.C. Preamplifier in a Grass Instruments Co., Model 7 Polygraph, Grass Instruments Co., Quincy, Massachusetts, USA). A continuous electrocardiogram was recorded using subcutaneous electrodes with an EKG-Pulse preamplifier in a Model 7 polygraph (Grass Instruments Co.). Heart rate was measured from the electrocardiogram.

An indwelling peritoneal catheter was placed through a midline incision 1 cm below the xiphoid process. A Tenckhoff type catheter was advanced into the peritoneal cavity with the tip in the right lower quadrant of the abdomen.

The animals were hydrated through the venous cannula with warmed lactated Ringers solution (Baxter–Travenol Company, Deerfield, Illinois, USA). The infusion rate was maintained at 8.8 to 13.3 ml/hr with a syringe pump (Sage Instruments, Model 341). Peritoneal exchanges were not begun until each rat was undergoing diuresis (90 to 120 min after the i.v. infusion was begun). The infusion rate was chosen to replace fluid losses due to peritoneal ultrafiltration (UF), and to maintain the rats in an expanded, diuretic state.

Dialysis solutions. The peritoneal dialysis solution used was Travenol Dianeal PD-2 containing sodium 132 mEq/liter, calcium 3.5 mEq/liter, magnesium 0.5 mEq/liter, chloride 96 mEq/liter, lactate 40 mEq/liter and 1.5% or 4.25% monohydrous dextrose. The actual anhydrous glucose concentrations in these solutions are 1.36 and 3.86%. Additional dextrose was added to the solutions for exchanges with higher dextrose concentrations. Anhydrous powder was added to achieve total anhydrous concentrations as shown. All dialysis solutions were adjusted to a pH of 7.4 with 1.0 N sodium hydroxide.

Peritoneal dialysis protocol. After commencement of diuresis, blood was drawn from the tail vein to obtain serum osmolality and hematocrit prior to any exchanges. After a five minute wash–out with 1.5% Dianeal (pH 7.4), 16 ml of dialysis solution with dextrose concentration as per protocol were infused into the peritoneum in approximately 15 seconds. The solution was allowed to dwell in the peritoneal cavity for 25 minutes, after which drainage was performed over a five minute period. The total cycle time was 30 minutes. Drainage was by gravity only, with slight manual agitation of the abdomen. Sacrifice and direct aspiration of residual fluid have revealed residual volumes near 1 ml with this technique. Since even very large molecular size volume markers are absorbed by rat lymphatics [6], mechanical drainage may be as accurate. At the end of each exchange, blood hematocrit was measured.

Protocols consisted of a series of exchanges (usually 4 to 7) with varying dextrose concentrations. Exchanges were repeated with progressive increments in dextrose concentration in six rats. Fourteen rats underwent a series of exchanges at a fixed dialysis solution glucose concentration of 15% (10), 6% (2), 1.4% (2) respectively. Prior to exchanges, serum glucose concentration was measured using Chemstrip BG (Boehringer Mannheim Diagnostic, Inc., Indianapolis, Indiana, USA). Insulin was added to peritoneal dialysis solutions (0.1 units of pork insulin/g% dextrose) and intravenously as necessary to maintain serum glucose at 200 mg% or less.

Special studies. Six animals underwent exchanges with 15 g% dextrose at a reduced instillation volume of 10 ml. The purpose of this study was to assess the effects of instillation volume on maximum ultrafiltration rate.

In two rats dextrose concentrations were decreased from two exchanges with 20% dextrose to two exchanges with 3.9% dextrose.

Six rats had consecutive 15% dextrose exchanges with total cycle times of 15 and 25 minutes (10 and 15 min dwells); the order was alternated. These studies were to see if net UF was proportional to cycle time at the maximum rate.

Seven rats were exposed to 5, 10, 15, and 20% dextrose exchanges at 15 minute total cycle times (10 minute dwells) to see if a maximum net UF per exchange could be demonstrated at short cycle times and lower net UF volumes, but at the maximum rate of UF seen with longer cycles. In three rats, consecutive exchanges went from low to high dextrose; in four, from high to low.

Two animals underwent a series of 16 ml exchanges with 15 g% dextrose; after control exchanges, nitroprusside (4.5 mg/liter) was added to the instilled solution. Five animals underwent exchanges with 15 g% dextrose and 16 ml volumes; after control exchanges, norepinephrine was infused intravenously (0.3 μg/kg/min). Three animals underwent studies with 16 ml, 15 g% dextrose exchanges; after control exchanges, Dobutamine was instilled intravenously (5.0 μg/kg/min). For each animal, single mean values of ultrafiltration were calculated for control and for drug associated exchanges.

Laboratory instruments. Osmolalities were measured in dialysate solutions before and after each exchange and in serum samples as above, using a Wescor 5100 B vapor pressure osmometer (Wescor Inc., Logan, Utah, USA). Concentrations of sodium were determined with a flame photometer (Model 343, Instrumentation Laboratories, Inc., Lexington, Massachusetts, USA).

Histology. Following completion of the exchange protocol, animals were sacrificed and tissue samples were taken from the abdominal wall, intestine and mesentery, to see if mesothelial alterations secondary to the exposure to the hypertonic solutions were detectable. The tissues were prepared for light microscopy with "en face" silver staining and mounted as previously described [7].

Statistics and calculations

Net ultrafiltration per exchange was calculated as drainage volume minus instillation volume. In studies with cardiovascular drugs, mean values of net ultrafiltration per exchange, mean blood pressure, and mean heart rate with control and drug associated exchanges were compared by the paired Student's *t*-test. The mean osmotic gradient during an exchange (mOsm/kg H_2O) was calculated as the average of dialysate minus serum osmolality at the beginning and at the end of the exchange. Net sodium removal per liter of net ultrafiltration was related to serum sodium calculated as (sodium concentration in instilled solution × the instilled volume)–(sodium concentration in drainage × the drainage volume)/net ultrafiltration/mean serum sodium concentration from 10 control rats [8].

Results

Overall, mean serum osmolality in 19 rats at the beginning of experiments was 289 mOsm/kg H_2O ± 10 SEM, and mean serum osmolality at the end of experiments was 331 ± 31. This most likely reflects some rise in serum glucose and some increase in serum sodium concentration related to the net sodium sieving effects of peritoneal ultrafiltration [8, 9]. This small rise had little effect on the osmotic gradient. Mean hematocrit at the beginning of studies was 44 ± 0.6 (N = 24) and the end of studies was 45 ± 1.1 (N = 17). Heart rates and blood pressures were stable except for nitroprusside studies mentioned below.

Table 1 summarizes the effects of dextrose concentration in dialysis solution on net ultrafiltration per exchange. The Table shows the number of animals subjected to each type of exchange. In those animals undergoing more than one exchange with a given dextrose concentration, a single mean value per animal was used. Mean values for solutions above 8 g% dextrose suggest a plateau in ultrafiltration per exchange. Ultrafiltration rate based on total cycle time at maximum averaged 0.42 ml/min.

Figure 1 shows results from studies in six rats where there were progressive increases in dialysis solution dextrose–concentrations and mean osmotic gradients over a series of exchanges up to seven exchanges. Increases in net ultrafiltration (UF) per exchange are seen with increases in mean osmotic

Table 1. Effects of increasing dextrose concentration, (mean ± SEM)

Dextrose *g/dl*	*N* *animals*	Gradient[a] *mOsm/kg*	UF[b] *ml/exchange*
1.4	6	35 ± 3	2.4 ± 0.6
3.0	4	86 ± 10	5.3 ± 0.3
3.9	4	125 ± 6	6.2 ± 0.1
6.0	10	186 ± 10	7.4 ± 0.5
8.0	7	262 ± 15	9.9 ± 0.5
9.0	2	330 ± 8	11.0 ± 1.0
10.0	4	344 ± 32	10.6 ± 0.8
15.0	16	488 ± 12	12.2 ± 0.4
20.0	4	690 ± 13	12.0 ± 0.4

[a] Time averaged.
[b] Means using one value per rat, thus a mean of means with *N* and SEM based on number of rats.

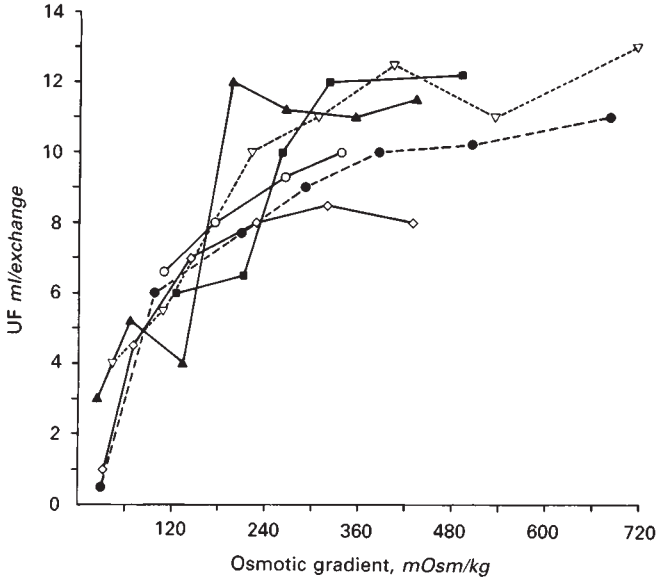

Fig. 1. *Ultrafiltration (UF) is related to the mean time averaged osmotic gradient during each exchange.* In these studies, progressive increases in dextrose were used during a series of exchanges. Symbols are rat number: (■) 543; (○) 544; (◇) 549; (▲) 550; (▽) 600; (●) 601.

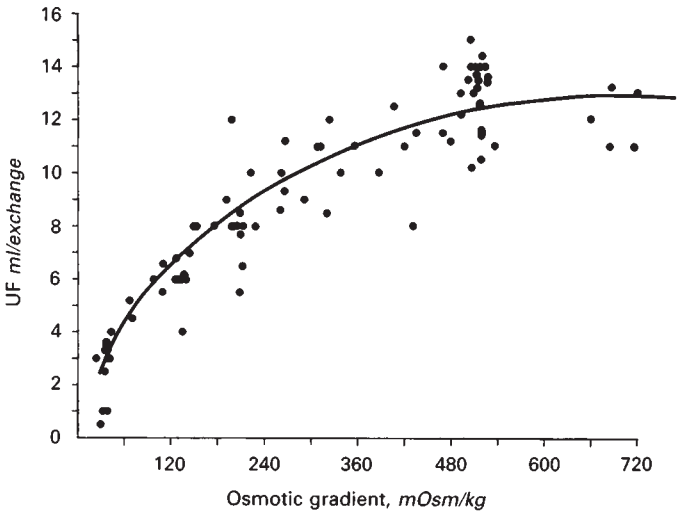

Fig. 2. *Mass plot of UF measurements from all exchanges using 16 ml instilled volumes without cardiovascular drugs are related to the mean time averaged osmotic gradient.*

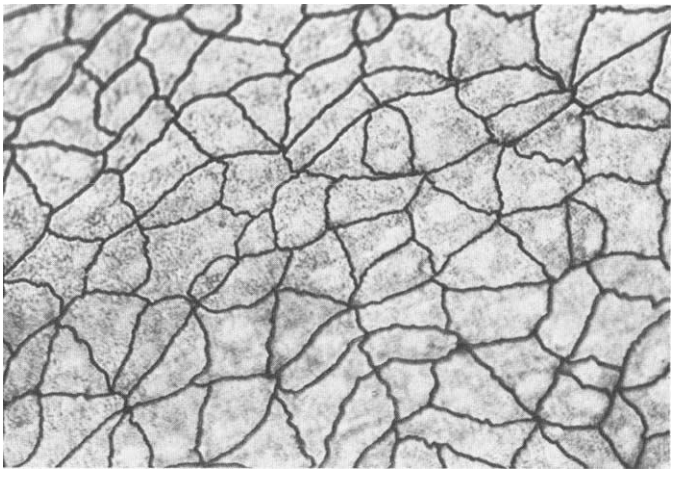

Fig. 3. *En face silver stain of visual mesentery from a rat exposed to 15% dextrose exchanges.* Mesothelial cell boundaries appear intact. (magnification 110×)

gradient to 240 mOsm/kg. Thereafter, there appears to be a plateau or reduction in the increments.

Figure 2 shows the mass plot of all net UF measurements per exchange in all rat studies. This includes studies from rats that underwent serial exchanges at the same or decreasing dextrose concentrations in dialysis solution for consecutive exchanges. Points fall close to the same ranges of UF per exchange at any given osmotic gradient as in Figure 1. In any single rat experiment, net UF per exchange usually fell in the same range for given dextrose concentrations.

Figure 3 shows the typical appearance of mesentery in rats subjected to these experiments. Mesothelial boundaries were still intact, no mesothelial denudation or injury was apparent. As net ultrafiltration per exchange increased, above 12 ml/exchange, the net removal of water was accompanied by relatively low amounts of sodium compared to extracellular fluid and yielded ratios of net sodium removal per liter of UF over serum sodium concentrations of 0.85 or less. This sieving effect has been well recognized as characteristic of peritoneal ultrafiltration and is maintained under the conditions of these studies [9].

Table 2. Effect of V_{IN} at dextrose, 15 g/dl (mean ± SEM)

V_{in} *ml*	*N* *animals*	UF *ml/exchange*	UF/V_{in}
16	15	12.5 ± 0.3	0.78 ± 0.02
10	6	8.0 ± 0.7	0.80 ± 0.07

V_{in} instilled volume

Table 2 shows the results in animals subjected to exchanges at 15 g% dextrose with 10 ml instillation volumes, as compared to those in animals with 16 ml instillation volumes. The ratio of net ultrafiltration to the instilled volume remains unchanged. Similar high mean time averaged osmotic gradients were main-

Table 3. UF with shorter cycle times (mean ± SEM)

Cycle time *min*	Instilled dextrose *g%*	UF *ml/exchange*	UF rate *ml/min*	*N animals*
Varied short cycle times				
15	15	7.6 ± 1.2	0.51	6
25	15	10.9 ± 1.2	0.43	6
Varied dextrose at fixed short cycle time				
15	5	3.5 ± 0.7	0.23	7
15	10	5.6 ± 0.4	0.37	7
15	15	6.7 ± 0.6	0.45	7
15	20	5.6 ± 0.5	0.37	7

Table 4. Effects of vasoactive agents at dextrose, 15 g/dl

Agent	Concentration	*N animals*	Mean UF *ml exchange* ± SEM Control	With agent
Nitroprusside	4.5 mg/liter (i.p.)	2	12.9 ± 0.1	12.4 ± 0.9
Norepinephrine	0.3 mcg/kg/min (i.v.)	5	13.5 ± 0.2	12.9 ± 0.5
Dobutamine	5.0 mcg/kg/min (i.v.)	3	13.0 ± 1.0	12.0 ± 1.2

tained (490 ± 8 and 488 ± 12 mOsm/kg H_2O with 10 ml and 16 ml exchanges, respectively); however, there may have been less fluid membrane contact proportional to volume as suggested by the fixed ratio.

When shorter cycles were used, there was also a plateau of UF rates (Table 3). At dextrose concentrations of 10% or higher, UF ranged from 0.37 to 0.51 ml/min (mean 0.43) which is comparable to that achieved with the longer cycles.

With 15% dextrose exchanges, UF rates were not significantly different with 15′ and 25′ cycles in the paired studies.

Table 4 shows the results in animals subjected to a series of exchanges with 15 g% dextrose while receiving i.p. or i.v. agents selected to alter peritoneal capillary blood–flow. Mean values from the drug studies showed no significant differences from mean values of ultrafiltration per exchange at the same dextrose concentration without drug exposure. There were no significant changes with drug administration except for a significant increase in heart rate with nitroprusside from 421 to 443 ($P < 0.01$). Other transient alterations in heart rate and blood pressure included bradycardia with norepinephrine, increased blood pressure with norepinephrine, and lowered blood pressure with nitroprusside.

Discussion

Our findings demonstrate a net ultrafiltration maximum at high osmotic pressure gradients in rats undergoing peritoneal dialysis cycles. Additional studies will be needed to determine if this represents filtration pressure equilibrium.

We have previously reviewed evidence that most of the peritoneal ultrafiltration represents an ultrafiltrate of peritoneal capillary blood [2–4]. However, recently proposed models of peritoneal transport emphasize the complexities related to solute and water distribution in tissues [10, 11]. Possible participation of lymphatics cannot be excluded [6, 10, 11].

Any lymphatic absorption would reduce net ultrafiltration and actual transcapillary ultrafiltration may be underestimated. Increases in lymphatic reabsorption rates could be important in limiting maximum net ultrafiltration.

It seems unlikely that increases in peritoneal fluid hydraulic pressure explain the limitations on ultrafiltration per exchange that we observed. Net ultrafiltration per exchange was proportionally reduced with low instillation volumes. With low volumes, mean osmotic gradient was still very high throughout the exchange and the reduced net ultrafiltration might represent primarily–reduced fluid membrane contact and reduced membrane surface exposed to the osmotic gradient. Solute clearances have been previously reported to be proportional to instillation volume [12]. If i.p. hydraulic pressure had significantly limited ultrafiltration per exchange at higher volumes, it would seem reasonable that the ratio of net ultrafiltration to instilled volume would not remain constant, but increase, as the i.p. hydraulic pressure was maintained lower. Also, the rats did not appear to develop tense ascites, not did changes in their vital signs suggest hemodynamic alternations that might be anticipated with respiratory compromise. Perhaps even more importantly, similar maximum UF rates were seen with shorter cycles and lower intraperitoneal volumes.

We cannot quantitate the possible magnitude of concentration polarization effects in the rat peritoneal circulation, but such effects must be considered [13].

Accurate assessment of osmotic pressures during peritoneal dialysis is not possible. The reflection coefficient for glucose has been indirectly measured in humans at 0.38 [14].

The average maximum ultrafiltration per 30 minute exchange in the rat was 12.5 ml, which averages over the exchange to be 0.42 ml/min. Instantaneous ultrafiltration rates would be highest at the beginning of the exchange and decreases due to glucose absorption [15]. However, with 30 minute cycles at very high osmotic gradients, ultrafiltration rate was relatively fixed when the osmotic gradient was maintained above that needed to induce maximum ultrafiltration. If 0.42 ml/min is the maximum average ultrafiltration rate possible with these cycle times and volumes due to filtration pressure equilibrium, and if indeed maximum filtration fraction is near 50%, as predicted by Ronco and colleagues [5], then effective capillary plasma–flow would be in the range of 0.84 ml/min. Using an average end–experiment hematocrit value of 45%, effective blood flow would be calculated as 0.84/0.55 = 1.5 ml/min. Such calculations of blood flow might be erroneous if sieving of electrolytes at the membrane interfaces exaggerates concentration polarization. In other studies, using 1.5% dextrose solutions yielding low ultrafiltration (1.2 ml/exchange), mean urea clearances were found to be 0.45 ± 0.01 SEM ml/min in 15 exchanges from five rats [16]. CO_2 diffusion into peritoneal dialysis solutions in humans [3] and hydrogen gas absorption from peritoneal dialysis solutions in rabbits [17] have suggested that mass–transfer area coefficients for these gases are estimates of effective blood flow and are two to three times urea clearances. Predictions of effective blood flow in our rat model as two to three times urea clearance measurements would predict 0.90 to 1.35 ml/min. The high value is near that estimated above from principles of filtration pressure equilibrium (1.5 ml/min).

There is no evidence that the very hypertonic exchanges caused major morphological or functional changes in the peritoneal membrane. Net ultrafiltration per exchange could be maintained at high levels, and decreases in osmotic gradient

with subsequent exchanges caused decreases in UF as expected. The sodium sieving effects support the integrity of the peritoneal membrane. We have previously explored possible mechanisms for electrolyte sieving during peritoneal dialysis [8, 9, 18]. None of these mechanisms would be expected to persist in the face of an injured membrane.

Perhaps most disconcerting to the filtration pressure equilibrium hypothesis is the lack of effect of drugs which were chosen to manipulate effective peritoneal capillary–flow. However, we have no direct proof that drugs changed effective peritoneal capillary–flow. Hypertonicity itself has been shown to be vasodilatory, as has lactate [4, 19–22]. There is good evidence that nitroprusside causes increases in venular permeability at the doses used, but it may not induce any greater arteriolar vasodilation of the microcirculation than the solutions themselves [4, 19, 21]. Increases in venular permeability might alter the reflection coefficient for glucose and reduce the effective osmotic pressure. Norepinephrine was used at a dose previously shown to reduce urea clearances [23]; since urea clearances are not usually thought to be blood flow limited, this would represent substantial reductions in the effective capillary flow. However, another animal model was used.

In summary, our findings in vivo are compatible with the possibility that maximum ultrafiltration primarily reflects the limitations of effective peritoneal capillary blood–flow and filtration pressure equilibrium. Additional studies are required to confirm this.

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References

1. Brenner BM, Ichikawa I, Deen WM: Glomerular filtration, in *The Kidney*, edited by Brenner BM, Rector FC Jr. Philadelphia, Saunders, 1981, pp. 291–307
2. Nolph KD, Miller FN, Pyle WK, Popovich RP, Sorkin MI: An hypothesis to explain the ultrafiltration characteristics of peritoneal dialysis. *Kidney Int* 20:543–548, 1981
3. Nolph KD, Popovich RP, Ghods AJ, Twardowski Z: Determinants of low clearances of small solutes during peritoneal dialysis. *Kidney Int* 13:117–123, 1978
4. Nolph KD, Miller F, Rubin J, Popovich R: New directions in peritoneal dialysis concepts and applications. *Kidney Int* 18: S111–S116, 1980
5. Ronco C, Brendolan A, Bragantini L, Chiramonte S, Feriani M, Fabris A, Dell Aquila R, Milan M, La Greca G: Flusso ematico capillare effettivo nel sistema dialitico peritoneale, in *Dialysi Peritoneale*, edited by Lamperi S, Capelli G, Carozzi S. Milano, Wichtig Editore, 1985, pp. 21–28
6. Courtice FC, Steinbeck AW: Absorption of protein from the peritoneal cavity. *J Physiol* 114:336–355, 1951
7. Verger C, Luger A, Moore HL, Nolph KD: Acute changes in peritoneal morphology and transport properties with infectious peritonitis and mechanical injury. *Kidney Int* 23:823–831, 1983
8. Nolph KD, Hano JE, Teschan PE: Peritoneal sodium transport during hypertonic peritoneal dialysis: Physiologic mechanisms and clinical implications. *Ann Int Med* 70:931–941, 1969
9. Ahearn DJ, Nolph KD: Controlled sodium removal with peritoneal dialysis. *Trans Am Soc Artif Organs* 28:423–428, 1972
10. Dedrick RL, Flessner MF, Collins JM, Schultz JC: Is the peritoneum a membrane? *ASAIO* 5:1–7, 1982
11. Flessner MF, Dedrick RL, Schultz JS: A distributed model of peritoneal–plasma transport: Theoretical consideration. *Am J Physiol* 246:R597–R607, 1984
12. Goldschmidt ZH, Pote HH, Katz MA, Shear L: Effect of dialysate volume on peritoneal kinetics. *Kidney Int* 5:240–245, 1975
13. Deen WM, Robertson CR, Brenner BM: Concentration polarization in an ultrafiltering capillary. *Biophysicol J* 14:412–431, 1974
14. Pyle WK, Popovich RP, Moncrief JW: Mass transfer evaluation in peritoneal dialysis, in *CAPD Update*, edited by Moncrief JW, Popovich RP. New York, Masson, 1981, pp. 35–52
15. Rubin J, Nolph KD, Popovich RP, Moncrief J, Prowant B: Drainage volumes during CAPD. *ASAIO* 2:2, 1979
16. Lal SM, Khanna R, Hain H, Moore HL, Nolph KD: Enhancement of peritoneal clearance with captopril. (abstracts) *Am Soc Neph* 18:84A, 1985
17. Aune S: Transperitoneal exchange: II. Peritoneal blood flow estimated by hydrogen gas clearance. *Scand J Gastroenterol* 5:99, 1970
18. Nolph KD: Solute and water transport during peritoneal dialysis. *Perspectives in Peritoneal Dialysis* 1:4–8, 1983
19. Miller FN, Nolph KD, Harris PD, Rubin J, Wiegman DL, Joshua IG, Twardowski ZJ, Ghods AJ: Comparison of the effects of altered peritoneal dialysis solutions on the microcirculation of the rat and on peritoneal clearances in man. *Kidney Int* 15:630–639, 1979
20. Janchem JR, Miller FN, Joshua IA, Weigman DL, Nolph KD, Rubin J, Harris PD: Mechanisms of dilation due to peritoneal dialysis solutions (abstract) *Physiologist* 21:59, 1978
21. Nolph KD, Ghods AJ, Brown P, Miller F, Harris P, Pyle K, Popovich R: Effects of nitroprusside on peritoneal mass transfer coefficients and microvascular physiology. *Trans Am Soc Artif Intern Organs* 23:210–218, 1977
22. Henderson LW, Nolph KD: Altered permeability of the peritoneal membrane after using hypertonic peritoneal dialysis fluid. *J Clin Invest* 48:992–1001, 1969
23. Hirszel P, Lasrich M, Maher JF: Augmentation of peritoneal mass transport by dopamine. Comparison with norepinephrine and evaluation of phamacologic mechanisms. *J Lab Clin Med* 94:747, 1979